RESEARCH ARTICLE

Genotyping of Peroxisome Proliferator-Activated Receptor gamma in Iranian Patients with *Helicobacter pylori* Infection

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Abstract

***Helicobacter pylori* (*H. pylori*) infection as a serious problem in both adults and children can induce chronic gastritis, peptic ulcer disease (PUD), and possibly gastric cancer. The aim of the current study was to survey antibiotic resistance and also to determine influence of PPARγ polymorphism in patients with *H. pylori* infection. During an 11-month-period, 98 *H. pylori* isolates were collected from 104 biopsy specimens. *In vitro* susceptibility of *H. pylori* isolates to 4 antimicrobial agents metronidazole, clarithromycin, amoxicillin and tetracycline were assessed by quantitative method according to European Committee on Antimicrobial Susceptibility Testing (EUCAST) guideline. PPARγ polymorphism was determined using polymerase chain reaction-restriction fragment length polymorphism assay. The frequency of *H. pylori* infection in our study was 94.2%. *In vitro* susceptibility data showed that highest level of resistance was related to metronidazole (66.3%), and the majority of *H. pylori* isolates were highly susceptible to amoxicillin and tetracycline (94.9% and 96.9%, respectively). Genotypic frequencies were 25.5% for CC (Pro12Pro), 40.8% for GC (Pro12Ala) and 33.7% for GG (Ala12Ala). In our study, CG genotype had highest distributions among infected patients with *H. pylori*. The study suggests that the PPAR-γ Pro12Ala polymorphism could be evaluated as a potential genetic marker for susceptibility to gastric cancer in the presence of *H. pylori* infection.**

Keywords: Peroxisome proliferator-activated receptor γ - *Helicobacter pylori* - gastric cancer

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Introduction

Helicobacter pylori (*H. pylori*) is a gram-negative, rod-shaped, flagellate, microaerophilic spiral bacillus and one of common bacterial infections in the world that colonizes the stomachs of about 50-60% of the world's population (Parsonnet et al., 1991). Infection with the bacteria is important public health problems in developing countries as well as in developed countries (Frenck Jr and Clemens, 2003). Person-to-person contacts, oral-oral and fecal-oral routes are major routes of transmission. Effective antibacterial therapy in order to eradication of infection is necessary. Combination therapy consisting of a proton pump inhibitor with a macrolide and a β-lactam as an eradication regiment could be effective for treatment of *H. pylori* infections, but antimicrobial resistance has been reported by several investigators (Eun et al., 2003). *H. pylori* is responsible for a spectrum of infections that can be ranged from mild or chronic gastritis to peptic ulcer, gastric lymphoma, and gastric cancer (Vilaichone et al., 2014).

Gastric cancer is the second most common cancer in the world and long-standing infection with *H. pylori* is linked to gastric cancer. However, exact mechanism that underlying to *H. pylori*-associated gastric carcinogenesis is still poorly understood (Uemura et al., 2001, Eun et al., 2003). It is clear that the pathogenicity of *H. pylori* is not only explained by bacterial virulence factors alone. Recently, several researchers have proved the role of host genetic factors involved in the *H. pylori*-associated gastric carcinogenesis (Tahara et al., 2008; Shibata et al., 2010; Goto et al., 2011; Jing et al., 2012).

The peroxisome proliferator-activated receptors (PPARs), members of the nuclear receptor superfamily, are ligand-dependent transcription factor that play an important role in cellular differentiation and carcinogenesis as well as regulation of fatty acid oxidation and glucose utilization. To date, three different PPAR subtypes (α, β, and γ) have been described (Michalik et al., 2004). Human PPARγ gene, located on chromosome 3, is consists of 9 exons (A1, A2, B, and 1-6), from which the two isoforms PPARγ1 and PPARγ2 are generated through alternative

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